A CASE OF DELUSIONAL PARASITOSIS ASSOCIATED WITH MULTIPLE LESIONS AT THE ROOT OF TRIGEMINAL NERVE

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ABSTRACT

The authors present a patient with multiple pontine lesions who exhibited symptoms consistent with delusional parasitosis. The trigeminal nerve nuclei are located throughout the brainstem. Pathology in either the nuclei or the branches of the fifth cranial nerve has been associated with both sensory and motor disturbances. Delusional parasitosis is a condition in which the patient has the firm belief that small, living organisms have infested his or her skin or other organs. To our knowledge, this is the first case report of delusional parasitosis associated with lesions at the root of the trigeminal nerve.

INTRODUCTION

All three divisions of the trigeminal nerve perform both motor functions (innervating the large muscles that contribute to mastication) and sensory functions (conveying stimuli from the face). The motor nucleus of the trigeminal nerve is located entirely in the pons. By comparison, the sensory nucleus is relatively diffuse and extends from the midbrain to the medulla. All three branches of the fifth cranial nerve exit the brainstem via the cerebellopontine angle.

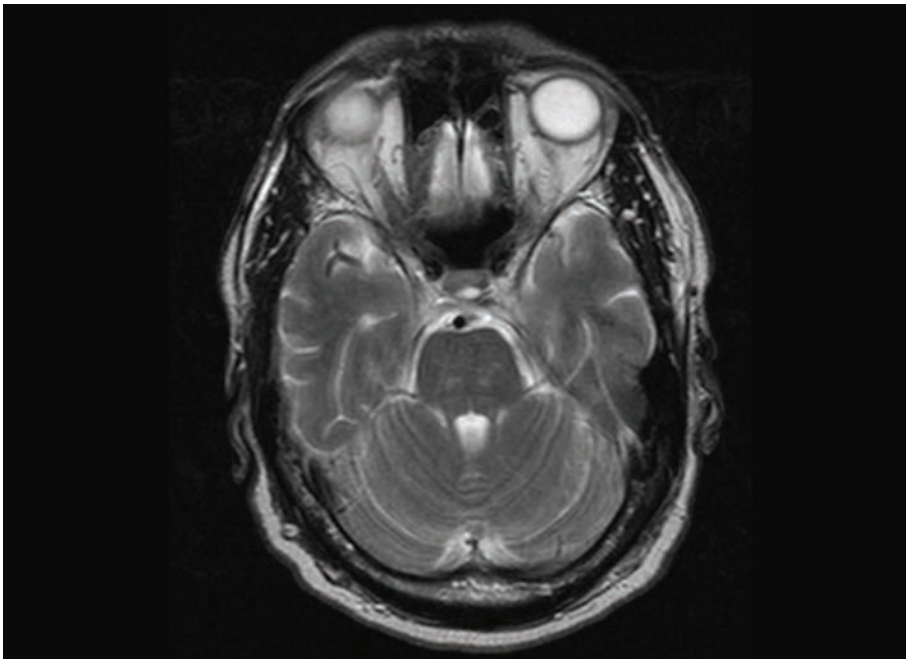

FIGURE 1. Axial cut at level of pons showing scattered foci of increased T2 signal

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Injury of the trigeminal nerve by either trauma or compression may lead to decreased function, including facial hypalgesia, afferent corneal reflex impairment, jaw jerk hypoactivity, and deviation of the jaw toward the side of lesion. Alternatively, irritation of the nerve may lead to trigeminal neuralgia (characterized by bursts of lacerating facial pain), as well as altered sensory perceptions.[1]

We present a case of delusional parasitosis that is likely the result of altered sensory perceptions associated with multiple pontine lesions at the root of the sensory division of the trigeminal nerve.

CASE REPORT

Chief complaint. A 65-year-old African American man presented to the outpatient mental health clinic complaining of worsening "infestation" of his face, mouth, and ears and thoughts of jabbing an ice pick in his ears to rid himself of the parasites.

History of present illness. The patient had no prior psychiatric history until seven years prior to presentation (at age 58), when he began complaining of "bugs" inhabiting the tissue of his cheeks and moving throughout his mouth, nose, cheeks, and ears. He "knew" they were there because their movement caused tingling and stabbing pains, especially on the right side of his face. The patient's belief of infestation was present for several months after the sensations began and were consistently unshakeable throughout his treatment. He reported both "feeling" and "seeing" the bugs and submitted "specimens." He attempted various methods to remove the "bugs," including intranasal Flonase® use (GlaxoSmithKline, Research Triangle Park, North Carolina), swishing diluted beach, leaning over an open flame in an attempt to "burn the critters," and pouring peroxide and alcohol into his ears. He also applied Raid® (S. C. Johnson & Son, Inc., Brantford, Ontario) in his ears and "smoked" cigarettes by lighting the filtered end and placing them in his ears in an attempt to fumigate the "insects." Additionally, he used cocaine to "numb" the bugs by either snorting or applying directly to the oral cavity. He stated that using cocaine directly on the oral mucosa temporarily relieved the tingling sensation and pain. The patient denied experiencing depressive, manic, or anxiety symptoms, and these symptoms were not observed objectively. He denied memory, concentration, or executive function deficits (confirmed via bedside testing). He denied experiencing other firm beliefs, paranoia, ideas of reference, hallucinations, or impairments in his thought processes.

Relevant medical history included a traumatic right facial laceration suffered at age five when he was ejected through the windshield of a car during an accident. He denied losing consciousness and denied a history of head injuries or seizures. Healing of the facial lacerations was complicated by keloid formation. This was treated aggressively over the course of his lifetime with injections, radiation, and multiple surgical revisions. The last of these treatments was a surgical revision that occurred about a decade before the patient was deployed to Vietnam. The patient lived alone, was unemployed, and was estranged from his long-term girlfriend and daughter. He was a retired Army officer. He served time in prison for cocaine possession. He denied current alcohol, tobacco, and other illicit substance use. He denied prescription medication use and over-the-counter/herbal product use.

During the initial interview, the patient was cooperative and engaging. He interacted politely and calmly with the examiners. His appearance was well groomed with appropriate eye contact. There was no evidence of increased or decreased psychomotor activity. His speech was slightly slow in rate but easily understood with appropriate prosody. He had a tendency to become animated when discussing the "bugs" but was able to lower his vocal tone with redirection. He described his mood as frustrated, and his emotional responses were appropriate to the topics of discussion. There was no evidence of lability. He had no deficits in attention, orientation, memory, or concentration. His thought processes were clear, coherent, and goal-directed. While discussing most topics, he was consistently logical. However, he tended to perseverate when discussing the "bugs" and occasionally required redirection to which he easily responded. With the exception of his infestation belief, there was no other evidence of disturbed thought content. He denied experiencing auditory and visual hallucinations. He described tactile hallucinations of the "bugs" moving around and the resulting subsequent burning sensation from their movement. Other than expressing frustration that his providers doubted the existence of the "bugs," he was without paranoia. He denied other delusions except that of the parasites. He denied thoughts of harming others, but had ongoing thoughts of stabbing himself with an ice pick to rid himself of the parasites (without intent to end his life). He had limited insight into his condition, as evidenced by his insistence that the bugs must exist if he was able to feel their movement, despite evidence to the contrary. His judgment was poor as evidenced by his use of various harmful methods to "exterminate the bugs." His Mini Mental State Examination (MMSE) score was 27 and bedside testing of executive functioning did not show signs of impairment [The Executive Interview (EXIT25©) score 8]. Due to his nonadherence, formal neuropsychological testing could not be arranged.

Treatment course. The patient had an in-depth exam into the etiology of his complaints. There were no physical exam findings suggestive of trigeminal nerve damage. There were no noted abnormalities in his gait and no other

cranial nerve abnormalities noted in previous physical exams. Laboratory studies, including complete blood count (CBC), chemistries, human immunodeficiency virus (HIV), hepatitis C virus (HCV), and rapid plasma reagin (RPR), were either negative or within normal limits. Several ear, nose, and throat (ENT) consults were obtained, none of which found evidence of infestation. He brought in several "specimens," which were determined on pathological exam to be skin cells and cerumen. A head computed tomography (CT) scan and magnetic resonance imaging (MRI) were obtained. The head CT was unremarkable; but the MRI revealed several scattered foci of increased T2 and fluid-attenuated inversion recovery (FLAIR) signal in the periventricular and deep white matter of the bilateral cerebral hemispheres (Figure 1). Similar appearing lesions were noted in the paramedian pons bilaterally.

The patient was treated with several antipsychotics with little success. Pimozide was discontinued secondary to prolonged QT interval, risperidone was discontinued due to akathisia, and quetiapine was discontinued secondary to ineffectiveness. He completed a 28-day rehabiliation program for cocaine use, and had several months of sobriety. However, increasing facial pain and tingling typically led to relapse of his cocaine use.

The patient was admitted to the inpatient psychiatric ward at the local Veterans Affairs several weeks after initial presentation for suicidal ideations in the context of "reaching the final straw" due to being unable to "exterminate the bugs." During this admission, he was started on gabapentin (to target the sensory perceptions via GABA agonist activity) and olanzapine (to target his delusion). His thoughts of self harm quickly resolved and he was discharged with instructions to follow up with both psychiatry and neurology. He attended a subsequent appointment in psychiatry in which he reported a mild subjective response to gabapentin as evidence by self-reported decrease in the "bug movements." However, the belief of infestation remained. He did not keep subsequent follow-up appointments with mental health and never kept his initial appointment with neurology.

DISCUSSION

Delusional disorder is characterized by the maintenance of nonbizarre, fixed, false beliefs not attributable to other psychiatric disorders for at least one month and not consistent with known cultural or religious beliefs. The prevalence in the United States is approximately 0.025 to 0.030 percent. There are various subtypes of delusional disorders, including jealous type, erotomanic type, and somatic type. Delusional parasitosis, or the delusion of infestation, is classified as a somatic type delusion.[2]

In 1938, Ekbom[3] presented a series of cases in which patients considered themselves to be infested with parasites when they were not. He theorized that abnormal sensations in these patients led to the delusion that parasites were present. He supported this notion by recognizing that other signs of psychosis were absent, such as ideas of reference or disturbances of thought outside of those involving infestation. Furthermore, he theorized that the patients were experiencing real perceptions rather than hallucinations and then applying a delusional explanation for them. He gave evidence supporting his claims, including that the patients' symptoms were concrete, localized, and were relieved temporarily by methods, such as scratching. Additionally, while some patients' explanations for their sensations changed with time, the sensations themselves did not.[3]

This theory was reiterated decades later by several authors, including Berrios,[4] who speculated that patients who experience tactile sensations gradually switch from perceiving their sensations "as if" insects were crawling on them to believing that they were definitely crawling on them. When later discussing how it is that some patients "see" skin cells, fibers, and hairs as insects as well as feeling them, Slaughter et al[5] theorized that this visual misperception resulted from the power of autosuggestion.

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Other authors have examined the association between the brain stem and psychosis, including delusional parasitosis. In 1975, Fisman[6] noted upon post-mortem pathological exam that patients with schizophrenia were more likely than their nonschizophrenic counterparts to have glial knots and perivascular infiltration in the region of the fifth nerve nucleus. More recently, Hanihara et al[7] described a case of delusional psychosis in a 63-year-old woman who had suffered a hemorrhagic stroke in the left posterior thalamus extending to the internal capsule and the head of the left caudate nucleus. She developed thalamic pain syndrome with burning sensations in her right hand and right half of her face. In the setting of these sensations, she developed the belief that live worms had infested her oral cavity. She later

responded to treatment, and the authors theorized that disconnected cortical sensory systems due to the stroke led to the development of tactile hallucinations.[7]

The presented patient's facial injury in childhood and subsequent keloid treatments likely caused right trigeminal nerve damage and subsequent paresthesias. These paresthesias could have then been misinterpreted by his cortex as infestation. The increased intensity of sensations on the right side of his face lends credence to this as a possible contributing factor. However, this cannot fully explain his sensations as he had them on both sides of his face. We hypothesize that his brain stem lesions could also have been at least associated with transmitting abnormal sensory input to his cerebral cortices, resulting in the development of his infestation belief (i.e., normal cortex interpreting abnormal sensory input from the trigeminal sensory root). These lesions were located in the pons bilaterally and corresponded with the location of the diffuse trigeminal nerve sensory nuclei. Perhaps these lesions could have led to the misinterpretation of the sensory input from the trigeminal nerves bilaterally (worse on the right than the left due to the childhood injury on the right side) into false sensations of tingling and pain on his face. These sensations were then in turn falsely attributed to "bugs" by his cortex. As such, gabapentin was initiated in a similar capacity to someone who suffers from trigeminal neuralgia in that the GABAeric agent would reduce the parathesias, thus possibly reducing the intensity of the infestation belief.

There are other possible etiologies for his current symptoms. Demyelination or other damage to the trigeminal nerves at the root entry zones in the pons can also cause trigeminal neuralgia. The patient could have had a genuine delusional disorder independent of the lesions in the pons and/or right trigeminal nerve damage. Additionally, the delusion could have been a part of another underlying mood or psychotic disorder that was not yet diagnosed. However, monitoring for signs of depression, mania, and other psychotic symptoms did not reveal objective evidence of mood or more severe psychotic disorders despite multiple opportunities for observation in both outpatient and inpatient settings. Although the patient experienced a decline in functioning as evidenced by estrangement from his girlfriend and daughter, jail time for drug conviction, and chronic unemployment, this decline was highly unlikely to be due to schizophrenia, given his ability to serve 20 years in the Army (and retire from it) without evidence of impairment during his 20s, 30s, and 40s; however, due to an inability to obtain historical corroboration from his girlfriend or daughter or have access to his military personnel records, this was difficult to determine. We opine that his decline in functioning was better explained by his cocaine use and the associated legal and social consequences. Of note, multiple interviews with the patient did not reveal significant religious or spiritual attitudes or beliefs regarding insects. He did not endorse prior traumatic experiences with bugs and did not assign a particular meaning to them.

The patient's age and gradual decline in functioning also raised concerns for a developing dementia. The patient's MRI revealed scattered foci of increased T2 and FLAIR signals in the periventricular and deep white matter of the bilateral cerebral cortices (Figure 1). It was possible that these lesions may have been the result of microvascular changes from the patient's episodic periods of cocaine-induced hypertension and small vessel dysfunction/constriction during his periods of cocaine intoxication, resulting in an early microvascular dementing process. This dementing process would have made it difficult for him to interpret abnormal perceptual experiences correctly or to have good judgment about the likely causes and cures of such perceptual experiences (resulting in development of this delusion later in life). However, during inpatient and outpatient observations, he displayed no impairment in memory and did not display evidence of aphasias, apraxias, agnosias, or impaired executive function (via bedside testing). Formal neuropsychiatric testing was not conducted. This does not exclude the possibility of a dementia, but it is difficult to make this determination without further follow-up visits.

The patient's cocaine use could have also resulted in the parathesias and tactile hallucinations, but he described experiencing paresthesias and tactile hallucinations prior to using cocaine (the veracity of this claim could not be confirmed). Additionally, he continued to experience the sensations and endorse the infestation belief while he was hospitalized in the VA inpatient psychiatric unit where he did not have access to cocaine. It should be noted that the patient found temporary relief when cocaine was applied directly to the oral mucosa. He may have received benefit from the anesthetic properties of cocaine. If so, his case might best be explained as abnormal sensations that were incorrectly interpreted as infestation, thus supporting the authors' theories.

Finally, the etiology of the patient's pontine lesions was unclear. Given that they also occurred in the cerebral hemispheres, they may have been related to ischemic events due to either an early vascular dementia or the patient's cocaine use. However, we do not have prior imaging with which to compare and establish a baseline. The authors also acknowledge that the lesions may have been coincidental and clinically silent (i.e., the small hyperlucencies in his age group may be normal findings).

Ultimately, confirmation of our theory or other etiologies is not possible as the patient was not adherent to follow-up appointments. To the authors' knowledge, this is the

first reported case of delusional parasitosis associated with prior trigeminal nerve damage and lesions in the root of the trigeminal nerve.

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REFERENCES

1. Kaufman DA. *Clinical Neurology for Psychiatrists*. Sixth Edition. Philadelphia, PA: Saunders Elsevier; 2007:34–35.
2. Kaplan JS, Saddock VA. *Synopsis of Psychiatry*. Tenth Edition. Philadelphia, PA: Lippincott Williams and Wilkins; 2007:504–510.
3. Ekbom KA, Yorston G, Miesch M, et al. The pre-senile delusion of infestation. *Hist Psychiatry*. 2003;14:232–256.
4. Berrios GE. Delusional parasitosis and physical disease. *Compr Psychiatry*. 1985;26:395–402.
5. Slaughter JR, Zanol K, Rezvani H, et al. Psychogenic parasitosis: a case series and literature review. *Psychosomatics*. 1998;39:491–500.
6. Fisman M. The brain stem in psychosis. *Brit J Psychiat*. 1975;126:414–422.
7. Hanihara T, Takahasi T, Washizuka, S, et al. Delusion of oral parasitosis and thalamic pain syndrome. *Psychosomatics*. 2009; 50:534–537.